Polymerase chain reaction diagnosis of Kingella kingae arthritis in a young child

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Table 1. Characteristics of liver transplant recipients with disseminated toxoplasmosis.

Reference/ age (y)/sex	Symptom onset (days posttreatment)	Source and method of diagnosis	Recipient/ donor serology	Type of infection	Treatment	Coinfection	Initial immunosuppressive treatment	Rejection	Outcome
[3] 24/F	Fever, pneumonia (postoperative)	Autopsy, routine staining	NA	Reactivation?	None	*Pseudomonas aeruginosa,* fungal?	Aza, P	No	Died
[4] 15/F	Fever and pneumonia (14), encephalitis (30)	Serology, BAL (retrospective stain)	Neg/IgG+, IgM−	Primary	TMP-SMZ and Py	HSV-1	Antithymocyte antibody, Aza, P	No	Survived
[5] 30/F	Fever (24), pneumonia (24), multiorgan failure	Autopsy, routine staining	Neg/IgG+, IgM+	Primary	None	—	Antithymocyte globulin, Aza, P, CyA	Acute	Died
[6] Child/ NA	Fever, pneumonia (NA)	Autopsy	NA	NA	NA	NA	NA	NA	Died
[7] NA	Fever, pneumonia (27)	BAL, culture	NA	NA	NA	NA	Tacrolimus, MP	NA	Died
[PR] 53/F	Fever (125), multiorgan failure	BAL, blood, liver biopsy, autopsy	IgG+, IgM−/neg	Reactivation	Py and Cm	—	MP, Aza, CyA	Acute, chronic	Died

NOTE. Aza = azathioprine; BAL = bronchoalveolar lavage; CyA = cyclosporin A; Cm = clindamycin; HSV-1 = herpes simplex virus type 1; MP = methylprednisolone; NA = not available; P = prednisone; PR = present case; Py = pyrimethamine; Su = sulfonamide; TMP-SMZ = trimethoprim-sulfamethoxazole; + = positive; − = negative.

toxoplasmosis. Additionally, a PCR assay for *Toxoplasma* species in whole blood may be useful.

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Polymerase Chain Reaction Diagnosis of *Kingella kingae* Arthritis in a Young Child

Kingella kingae is a fastidious, gram-negative bacillus that causes osteoarticular infections, predominantly in small children <5 years of age [1]. Recovery of the organism with use of conventional solid culture media is often unsuccessful [2]. We describe a case of culture-negative arthritis in a small child in whom *K. kingae* could be detected in joint fluid only with use of broad-spectrum PCR amplification. To our knowledge, this is the first report of identification of *K. kingae* using this method.

A 2-year-old boy presented to the hospital for evaluation of fever, limp, and a swollen knee of 1 day's duration. The patient appeared nontoxic, with a rectal temperature of 38.7°C. The right knee was moderately swollen, erythematous, and warm to touch. The erythrocyte sedimentation rate was 52 mm/h and the C-reactive protein level was 48 mg/L. Serum IgG antibody titers to *Borrelia burgdorferi* were borderline (1:256) on days 1 and 8 of

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hospitalization; IgM antibodies to *B. burgdorferi* were negative. Evaluation of a joint-fluid aspirate revealed 168,000/mm^3 WBCs (92% polymorphonuclear cells) and a negative gram stain. Culture with use of conventional solid and liquid media, as well as a PCR assay for *B. burgdorferi* DNA, were negative. Therapy was instituted with intravenous amoxicillin/clavulanic acid for 7 days followed by 21 days of oral therapy. Four weeks after symptom onset the physical examination findings were entirely normal.

A specimen of the initial joint fluid had been stored at −20°C for possible future analysis. After thawing and centrifugation, DNA extraction from the pellet and broad-range bacterial PCR amplification were performed as previously described [3]. Universal primers complementary to constant regions from a part of the gene coding for 16S rRNA were used, followed by direct sequencing of the amplicon. The 412-nucleotide sequence was 99.7% and 99.5%, similar to the two available sequences of *K. kingae* with accession numbers X74902 and M22517, respectively. The next most closely related reference sequence was *Neisseria canis,* with a similarity of only 93.8%. On the basis of the eubacterial PCR results, arthritis due to infection with *K. kingae* was retrospectively diagnosed.

Although there have been no more than 112 cases of infections due to *K. kingae* in children reported to date [4], this organism may occur more frequently than assumed. Cultivation can be improved by inoculating specimens directly into BACTEC blood culture bottles (Becton Dickinson, Sparks, MD) [5]; however, this procedure is not routine in many clinical laboratories. Reports from institutions where the organism is consistently sought, show it to be the second most common cause of infectious arthritis in children; *Staphylococcus aureus* is the most common cause [5].

PCR has been used to identify other poorly growing organisms known to cause infectious arthritis such as *B. burgdorferi* [6], *Neisseria gonorrhoeae* [7], and *Tropheryma whippelii* [8]. With the exception of *T. whippelii,* specific, rather than broad-range, amplification systems were employed for identification in these cases.

In addition to using standard solid media to recover usual pathogens, routine injection of specimens from osteoarticular infections into BACTEC media (Becton Dickinson) is the diagnostic method of choice to ensure adequate recovery of *K. kingae* [5]. We recommend broad-spectrum PCR amplification as a second-line technique to identify *K. kingae,* or other organisms, when use of culture media is unsuccessful.

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Shock and Cerebral Infarct After Rifampin Re-exposure in a Patient Infected with Human Immunodeficiency Virus

Adverse reactions after rifampin administration are uncommon; neurologic complications are rare, and episodes of anaphylactic shock are infrequent. We describe a patient who developed an extensive cerebral infarct, apparently because of severe hypotension after rifampin re-exposure. To our knowledge, such an adverse reaction has not been reported to date.

A 33-year-old man was admitted to our hospital for evaluation of fever and pulmonary infiltrates that proved to be caused by tuberculosis. A serology for antibodies to HIV was positive, and the patient's CD4 cell count was 214/μL. Therapy with a fixed-dose compound containing isoniazid (300 mg/d), rifampin (720 mg/d), and pyrazinamide (1800 mg/d) was instituted, and the patient was discharged 4 days later with improvement of his condition. He returned to the emergency department 6 days later with complaints of recurrence of fever and generalized myalgia. All drugs were administered in the emergency department under medical supervision. The patient underwent close observation during the following 6 hours, and no adverse effect was detected. A Jarisch-Herxheimer reaction was suspected and prednisone was added to the treatment regimen.

The patient returned 6 days later reporting the same symptoms in addition to rhinorrhea. Pyrazinamide therapy was discontinued and streptomycin and ethambutol were added to the regimen, but his symptoms continued, and, 5 days later, all drugs were discontinued to be reintroduced at home one at a time. He restarted

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